A Case of Idiopathic Severe Acute Pancreatitis following Cesarean Section Delivery

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Acute pancreatitis rarely occurs in the postpartum period. Furthermore, there are very few reports of it after cesarean section delivery. A 35-year-old woman presented with dyspnea and abdominal distension on the third day after cesarean section delivery. Under a suspicion of acute pancreatitis, she was initially managed with conservative treatment. However, she developed intra-abdominal fluid collections and gastric bleeding, which were managed with percutaneous drainage, endoscopic hemostasis, and angiographic embolization. She was discharged with good clinical recovery. Postpartum pancreatitis, especially after cesarean section, is rare; however, its management is not different from that for usual pancreatitis. (Korean J Gastroenterol 2016;68:161-165)

Key Words: Pancreatitis; Postpartum period; Cesarean section; Gastric varix; Stomach ulcer

INTRODUCTION

Acute pancreatitis is a sterile inflammation that progresses to development of systemic inflammatory response syndrome and multisystem organ failure. Knowledge of the etiology and pathogenesis of acute pancreatitis has significantly increased; however, there are several aspects that have not been fully described. There are some common causes of acute pancreatitis such as gallstones, alcohol consumption, and endoscopic retrograde cholangiopancreatography.

Compared to the number of reports related to these common causes, there are few reports on acute pancreatitis associated with pregnancy. However, the incidence of acute pancreatitis in pregnant populations is similar to that in non-pregnant populations, moreover, its causes in pregnant and non-pregnant subjects are similar. Ramin and Ramsey observed an incidence of acute pancreatitis during pregnancy of 19% in the first trimester, 26% in the second trimester, 53% in the third trimester, and 2% in the postpartum period. Therefore, acute pancreatitis rarely occurs in the postpartum period. Furthermore, there are few reports about acute pancreatitis occurring after cesarean section delivery. Herein, we report a case of acute pancreatitis following cesarean section delivery.

CASE REPORT

The subject, a 35-year-old woman, presented to the emer-
Emergency department with a three-day history of dyspnea and abdominal distention. She was referred from another hospital for further management. Six weeks previously, she was managed with ritodrine, and nifedipine due to preterm labor. Three days previously, she underwent full-term emergency cesarean section. Although there was bleeding after the cesarean section without hypovolemic shock, she did not need a transfusion. Uterine bleeding improved after uterine massage and administration of a drug to cause uterine contraction. One day after her cesarean section, she had dyspnea and abdominal distension.

She had been healthy previously and had not consumed alcohol. On review of systems, she complained of dyspnea and abdominal distension without abdominal pain. On physical examination, her abdomen was distended without tenderness, and breath sounds were decreased in both lower lung fields.

On admission to the emergency department, she was conscious and oriented. Her vital signs were as follows: blood pressure, 163/82 mmHg; heart rate 139 beats/minute; respiratory rate 46 breaths/minute, and body temperature 38.3°C. Her arterial blood gas had a pH of 7.511, a PaO2 of 53.0 mmHg, a PaCO2 of 24.9 mmHg, and a HCO3 concentration of 19.9 mmol/L.

Further investigation revealed a total leukocyte count of 34.7×10^3/µL (4.0-10.0×10^3/µL), hemoglobin level of 15.1 g/dL (12-16 g/dL), platelet count of 253×10^3/µL (130-400×10^3/µL), BUN concentration of 12 mg/dL (10-26 mg/dL), creatinine level of 0.4 mg/dL (0.7-1.4 mg/dL), calcium concentration of 6.0 mg/dL (8.8-10.5 mg/dL), amylase level of 465 IU/L (30-100 IU/L), lipase level of 768 IU/L (23-300 IU/L), CRP level of 15.10 mg/dL (0-0.5 mg/dL), total bilirubin level of 1.4 mg/dL (0.2-1.2 mg/dL), albumin level of 2.0 g/dL (3.3-5.2 g/dL), AST level of 38 IU/L (0-40 IU/L), ALT level of 9 IU/L (0-40 IU/L), ALP level of 68 IU/L (30-115 IU/L), and a GGT level of 6 IU/L (8-35 IU/L). An initial chest X-ray showed bilateral pleural effusion and pulmonary edema. Contrast-enhanced CT of the abdomen confirmed pancreatitis and revealed a diffuse swollen pancreas with peri-pancreatic and retro-peritoneal fluid collection. A gallstone was observed, but there was no evidence of pancreatic duct dilatation or necrosis (Fig. 1). She developed respiratory distress and was given a 6 L O2 facial mask. Pleural fluid was removed by two chest percutaneous drainage (PCD) catheters (right and left) over a period of six days. She was conservatively managed with intravenous broad-spectrum antibiotics, parenteral nutrition, and other supportive care in the intensive care unit. One week after admission, her total leukocyte count and C-reactive protein level began to decline. She had daily fever of more than 38°C, but the fever peak declined. She had abdominal distension without any other symptoms.

Two weeks after admission, her abdominal distension had worsened. Follow-up CT revealed a considerable amount of fluid in the peritoneal cavity and retroperitoneum (Fig. 2). Three abdominal PCD tubes were placed for intra-abdominal collections. We checked the tubes and changed tube posi-

![Fig. 1. CT reveals pancreatic swelling and abdominal fluid collection. (A) On axial CT scan, diffuse pancreatic swelling and slightly reduced parenchymal attenuation are seen, but no indication of pancreatic necrosis or duct dilatation. (B) On coronal CT scan, the peri-pancreatic area, retroperitoneal space, mesentery, and transverse mesocolon fluid collections are seen. In the omentum, mesentery, and peritoneum, diffuse infiltration is seen. (C) On axial CT scan, a small stone is seen in the gallbladder.](image-url)
tions repeatedly over a period of 22 days. A fever of more than 38°C continued throughout the three weeks following admission. Intravenous antibiotics were provided for one month followed by oral antibiotics for one week. The patient was discharged after a 31-day stay in hospital. She exhibited a good clinical recovery during her visit two weeks after discharge.

However, three weeks after that discharge she presented

Fig. 2. CT of the abdomen reveals a considerable amount of fluid. On coronal CT scan, considerable fluid collection is seen in the peritoneal cavity and retroperitoneum. Accumulation of contrast enhancement is seen in the wall of the fluid collections, in keeping with tissue reaction and abscess formation.

Fig. 3. (A) An exposed vessel is present in the high body greater curvature anterior wall side. (B) Successfully installed hemoclips are shown. (C) Splenic vein obliteration (white arrow) is suspected on CT scan. (D) Histoacryl injection to the gastric varix is seen on the posterior wall side of the high body greater curvature. (E) Locations of posterior gastric artery pseudoaneurysm (black arrow) and left gastric artery angiographic embolization sites are shown.
to the emergency department with hematemesis due to Dieulafoy’s ulcer, which was treated by endoscopic hemoclipping (Fig. 3A, B). Two weeks after that treatment, she visited the emergency department with gastric variceal bleeding caused by splenic vein obliteration (Fig. 3C). After undergoing endoscopic sclerosing treatment (Fig. 3D), she was discharged. Two weeks later, she presented to the emergency department with melena, and underwent angiographic embolization (Fig. 3E). Six months after the melena treatment she reported no further bleeding.

DISCUSSION

The incidence of acute pancreatitis in pregnancy differs among the studies reported. Eddy et al. observed an incidence of acute pancreatitis of 101 among 305,101 pregnancies (1 in 3,021) in the United States from 1992 to 2001. Ramin and Ramsey found that 43 of 147,197 pregnant women (1 in 3,333) had acute pancreatitis, but noted only one case of postpartum pancreatitis in the United States from 1983 to 1993. In that case, the cause of the pancreatitis was gallstone. Hernandez et al. reported that the incidence of acute pancreatitis in pregnancy was 21 in 93,440 (1 in 4,449) in the United States from 1996 to 2006, whereas Xu et al. showed that it was 36 in 34,292 (1 in 953) in China from 1991 to 2014. The incidence of acute pancreatitis in these pregnant populations are similar to those in general and non-pregnant populations. Compared to the incidence of pancreatitis during pregnancy, postpartum acute pancreatitis is rare. Previous to this case report, few cases of acute pancreatitis following cesarean section have been reported. During pregnancy, increased estrogen and progesterone levels induce gallbladder smooth muscle relaxation and biliary stasis, which increases the volume of the gallbladder and decreases the flow of bile and ejection fraction. Thus, gallstone is the most common cause of pancreatitis in pregnant populations.

The pathogenesis of acute pancreatitis is not fully understood. Opie hypothesized that gallstones impacted in the common channel (junction between the common bile duct and the pancreatic duct) obstruct the outflow of bile, provoke bile reflux into the pancreatic duct and induce pancreatitis. Pancreatic duct obstruction can activate a cascade of digestive enzymes leading to autodigestive injury which stimulates an inflammatory response. However, several findings argue against a role for bile reflux in the pathogenesis of gallstone pancreatitis; too short (< 7 mm) common channel, high pancreatic ductal pressure, and the observation that non-infected bile does not cause pancreatic injury.

White et al. reported that the incidence of postoperative pancreatitis is higher in biliary surgery, gastrectomy, splenectomy, and aortic graft surgery, but its cause is not known. They presumed that trauma to the pancreatic tissue and ducts or impairment of the blood supply precipitate pancreatitis.

In the present case, it seems less likely that gallstone presence is the cause of the subject’s pancreatitis, despite the presence of a gallstone, because liver function tests revealed nearly normal levels, except for mild hyperbilirubinemia (total bilirubin 1.4 mg/dL) and there was no bile duct dilatation. Although we could not exclude passed stone or microlithiasis as a cause of pancreatitis, we thought the possibility was low. In addition, there was bleeding after cesarean section without hypovolemic shock. We could not exclude ischemia as a cause of pancreatitis. However, it is unclear whether the ischemia induces pancreatitis.

Alcohol is not considered a likely cause of the subject’s pancreatitis because she denied alcohol use. Although hyperlipidemia, hyperparathyroidism, trauma, medication, and fatty liver are less common causes of acute pancreatitis in pregnancy, there was no candidate cause of acute pancreatitis in this subject. However, a triglyceride check was missed at the time of admission, but the triglyceride level was 221 mg/dL (normal 0-150 mg/dL) one week after admission. Although a mild to moderate elevation in triglyceride levels (175-870 mg/dL) and a rapid decrease within 72 hours of presentation are common in the early phase of acute pancreatitis of any etiology, we could not exclude triglyceride as a possible cause of the subject’s pancreatitis.

Treatment of postpartum acute pancreatitis is not different from that for usual acute pancreatitis. Conservative management consisting of bowel rest, intravenous fluid hydration, and analgesia is recommended. In the present case, PCD for multiple intra-abdominal fluid collection, endoscopic hemoclipping and angiographic embolization were necessary after initially implementing a conservative management approach.

In summary, we report a case of postpartum idiopathic
acute pancreatitis following cesarean section. Postpartum pancreatitis, especially after cesarean section, is rare, but its management is not different from that for usual acute pancreatitis.

REFERENCES